Treating The Symptom By Knowing The Disease

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Respiratory distress is the most common symptom at the end of life but is consistently mismanaged

- The standard of care for respiratory distress
- There is not a simple bandaid
- Mismanagement will make things worse
- Medications have side effects
- Masking will only live up to hospice stereotype, costing our companies resources that are better utilized elsewhere

Signs of Respiratory Distress

Respiratory Rate
- Bpm > 20
- BPM < 8

Decreased Lung Volume
- Constricted airways
-Obstructed airways

Fast shallow breathing
-Difficulty or absence of speech

Work of Breathing
- Use of accessory muscles
- Gasping
- Tripoding
- Purse lip breathing

Periods of Apnea
- Absence of breathe followed by gasping and tachycardia

Cause and Effect of Respiratory Distress

Hypoxemia
- SOB
- Pulmonary hypertension
- Fatigue
- Broncho-Constriction

Wheezeing
-Tightened chest

Hypercapnia
-Euphoria
-Somnolence
-Unconscious
-Respiratory failure

Small Lung Volumes
-Increased WOB
-SOB

Sputum Production
-Increased Hypoxia
-Lung infections
-Obstructive cough

Fluid overload
-Pulmonary edema
-Basilar cracks

Standard Hospital Respiratory Distress Symptom Management

Oxygen Therapy
- Nasal cannula
- Simple face mask
- Venturi mask
- NON-Rebreather mask

Opioids
- Morphine
- Hydrocodone
- Methadone

Broncho-Dilators
- Albuterol/Levalbuterol
- Brovana
- Advair/Symbiort

Anticholinergics
- Ipratropium Bromide
- Spiriva
- Atropine drops
- Scopolamine patch

Additional Treatments

Anxiety Medications
- Benzodiazepines
  - Xanax
  - Lorazepam

Diuretics
- Furosemide
- Torsemide
- Bumetanide

Steroids
- Prednisone
- Dexamethasone

Antibiotics
Pulmonary Rehab
Surgery
**End of Life Respiratory Distress is Based on Ventilation**

Respiratory Effort is Based on CO2 Expulsion

- Each individual patient has a specific Minute Volume (MV) that controls their CO2 levels
- MV is based on the patient’s Tidal Volume Exhaled (VTE) and Respiratory Rate (RR)
- Decreasing the VTE will increase the RR
- Decreasing the RR will increase VT

**Pathophysiology COPD**

Hypoxemia is a result of a VQ mismatch.
Consequent Hypoxia is a result of airway flow limitations, pulmonary hypertension, and systemic inflammation.
Exertion Hypoxia is a result of decreased lung volumes
Increased mucus production is due to impaired gas exchange and compromised mucociliary lining
Hypercapnia is the result of lungs tissues decreased elasticity, hindering the recoil needed to expel CO2
Anxiety associated with being “Air Starved” can cause psychosomatic respiratory distress
Late stage respiratory distress is secondary to severely diminished lung volumes

**Physiologic Based Treatments of COPD Related Respiratory Distress**

<table>
<thead>
<tr>
<th>Physiology</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broncho-Constriction</td>
<td>Broncho-Dilators</td>
</tr>
<tr>
<td>Chronic Hypoxia</td>
<td>Oxygen</td>
</tr>
<tr>
<td>Excessive Mucus Production</td>
<td>Anticholinergics</td>
</tr>
<tr>
<td>Airway Swelling</td>
<td>Corticosteroids</td>
</tr>
<tr>
<td>Hypercapnia (&lt; CO2)</td>
<td>BIPAP</td>
</tr>
<tr>
<td>Decreased Lung volumes</td>
<td>Opioids</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Benzodiazepines</td>
</tr>
<tr>
<td>Exertion Induced Hypoxia</td>
<td>Discontinuing Exertion</td>
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</tbody>
</table>

**Side Effects of Standard Interventions for COPD Related Respiratory Distress**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Side effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Broncho-Dilators</td>
<td>&lt; LOC &lt; ability to ventilate &lt; ability to cough effectively</td>
</tr>
<tr>
<td>Oxygen</td>
<td>&lt; LOC &lt; ability to ventilate &lt; ability to cough effectively</td>
</tr>
<tr>
<td>Anticholinergics</td>
<td>Changes Glue like secretions</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>Laryngeal spasm (cough)</td>
</tr>
<tr>
<td>BIPAP</td>
<td>Claustraphobia/Asthma</td>
</tr>
<tr>
<td>Opioids</td>
<td>Calcium &lt; LOC &lt; Ability to ventilate &lt; Ability to cough effectively</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td></td>
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</tbody>
</table>
Symptom Management of COPD

Physiologic Effect of NPPV

BIPAP
- Increased Tidal Volume (VT) in, Increase Tidal Volume Exhaled (VTE) out = CO2 removal
- Increasing the VT, decreases RR
- Decreases WOB
- Decreases accessory muscle fatigue

Pathophysiology of CHF

- Pressure and volume overload
- Loss of cardiac muscle
- High output failure
- Reduction in cardiac output

Effects of left ventricular (LV) insufficiency/failure
1) There is an intrinsic decrease in muscle contractility
2) Increased reload backup, resulting in pulmonary congestion and dyspnea.
3) Systemic blood pressure is often reduced, but there is an increased after load, which can further reduce cardiac output
4) Heart rate is generally increased as part of a compensatory mechanism. An increase in heart size, increasing wall tension and increasing myocardial oxygen consumption

Physiologic Based Treatments of CHF Related Respiratory Distress

Physiology
- Pulmonary Edema
- Hypoxemia
- Exertion Hypoxemia
- Basilar Infiltrates
- Respiratory Infections

Treatment
1) BIPAP
2) Diuretics
Oxygen
Discontinuing Exertion
Incentive Spirometry
Antibiotics

Symptom Management of CHF

Physiologic Effect of NPPV

BIPAP
- Increases intrathoracic pressure
- Decreasing cardiac output (CO)
- The Right Ventricle (RV) slows
- The Left Ventricle (LV) catches up
- Kidneys resume normal Glomerular Filtration Rate (GFR)
- Pulmonary edema is able reabsorb into the body

Pathophysiology of ALS

Amyotrophic Lateral Sclerosis

Cellular processes that occur after disease onset
- Mitochondrial dysfunction
- Protein aggregation
- Generation of free radicals
- Excitotoxicity
- Inflammation and apoptosis

For most patients the underlying cause is unknown.
ALS diaphragmatic paralysis

Chest X-Ray Showing elevated right hemidiaphragm

Physiologic Based Treatments of ALS Related Respiratory Distress

<table>
<thead>
<tr>
<th>Source</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaphragmatic paralysis</td>
<td>BIPAP/Ventilator</td>
</tr>
<tr>
<td>Subglottic saliva hangup</td>
<td>Anticholinergic</td>
</tr>
<tr>
<td>Ineffective cough</td>
<td>1)Scopolamine patch</td>
</tr>
<tr>
<td>Epiglottic malfunction</td>
<td>2) Atropine sublingual drops</td>
</tr>
<tr>
<td></td>
<td>1) Cough assist</td>
</tr>
<tr>
<td></td>
<td>2) Suction machine</td>
</tr>
<tr>
<td></td>
<td>1) Dietary modifications</td>
</tr>
<tr>
<td></td>
<td>2) Swallowing techniques</td>
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</tbody>
</table>

Side Effects of Standard, Appropriate and Inappropriate, Interventions for ALS Related Respiratory Distress

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIPAP/Ventilator</td>
<td>PS set to high or low</td>
</tr>
<tr>
<td>Anticholinergic’s</td>
<td>1) Barotrauma</td>
</tr>
<tr>
<td>Oxygen</td>
<td>2) Atelectatic basilar lung lobes</td>
</tr>
<tr>
<td>Bronchodilators</td>
<td>Over drying of mouth and airways</td>
</tr>
<tr>
<td>Cough Assist</td>
<td>No physiologic benefit</td>
</tr>
<tr>
<td></td>
<td>&gt; HR, Shakes, Anxiety</td>
</tr>
<tr>
<td></td>
<td>1) Respiratory distress</td>
</tr>
<tr>
<td></td>
<td>2) Barotrauma</td>
</tr>
</tbody>
</table>

Symptom Management of ALS

Physiologic Effect of Positive Pressure Ventilation

<table>
<thead>
<tr>
<th>Non-Invasive (NPPV)</th>
<th>Pressure Support (PS) (IPAP-EPAP = PS)</th>
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<tbody>
<tr>
<td>Invasive (IPPV)</td>
<td>Delta Pressure (^P) (PIP-PEEP = ^P)</td>
</tr>
</tbody>
</table>

- Diminishes basilar lung atelectasis
- Decreases tachypnea
- Decreases tachycardia
- Prolongs ability to be comfortably off machine
- Decreased work of breathing
- Decreases use of accessory muscles

References

- http://radiologymasterclass.co.uk/tutorials/chest/chest_pathology/chest_pathology_page8.html
- http://circheartfailure.ahajournals.org/content/4/6/677.full